DISSERTATION ON

"A STUDY ON CHRONIC OBSTRUCTIVE PULMONARY DISEASES"

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations
for the award of the degree of

M.D. IN GENERAL MEDICINE

BRANCH – I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that this dissertation entitled "**"STUDY ON CHRONIC OBSTRUCTIVE PULMONARY DISEASES** is the bonafide original work of **Dr. SUGUMAR T** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu Dr.M.G.R. Medical University to be held in APRIL - 2016. The period of the study was from January – 2015 to August -2015.

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DECLARATION

I, **Dr.SUGUMAR T,** solemnly declare that dissertation titled **"“STUDY ON CHRONIC OBSTRUCTIVE PULMONARY DISEASES”** is a bonafide work done by me at Thanjavur Medical College and Hospital during January 2015 to August 2015 under guidance and supervision of my unit chief **Prof.Dr.K.NAGARAJAN, M.D.,** Professor and head of the Department of Medicine.

This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

Place: Thanjavur
Date:

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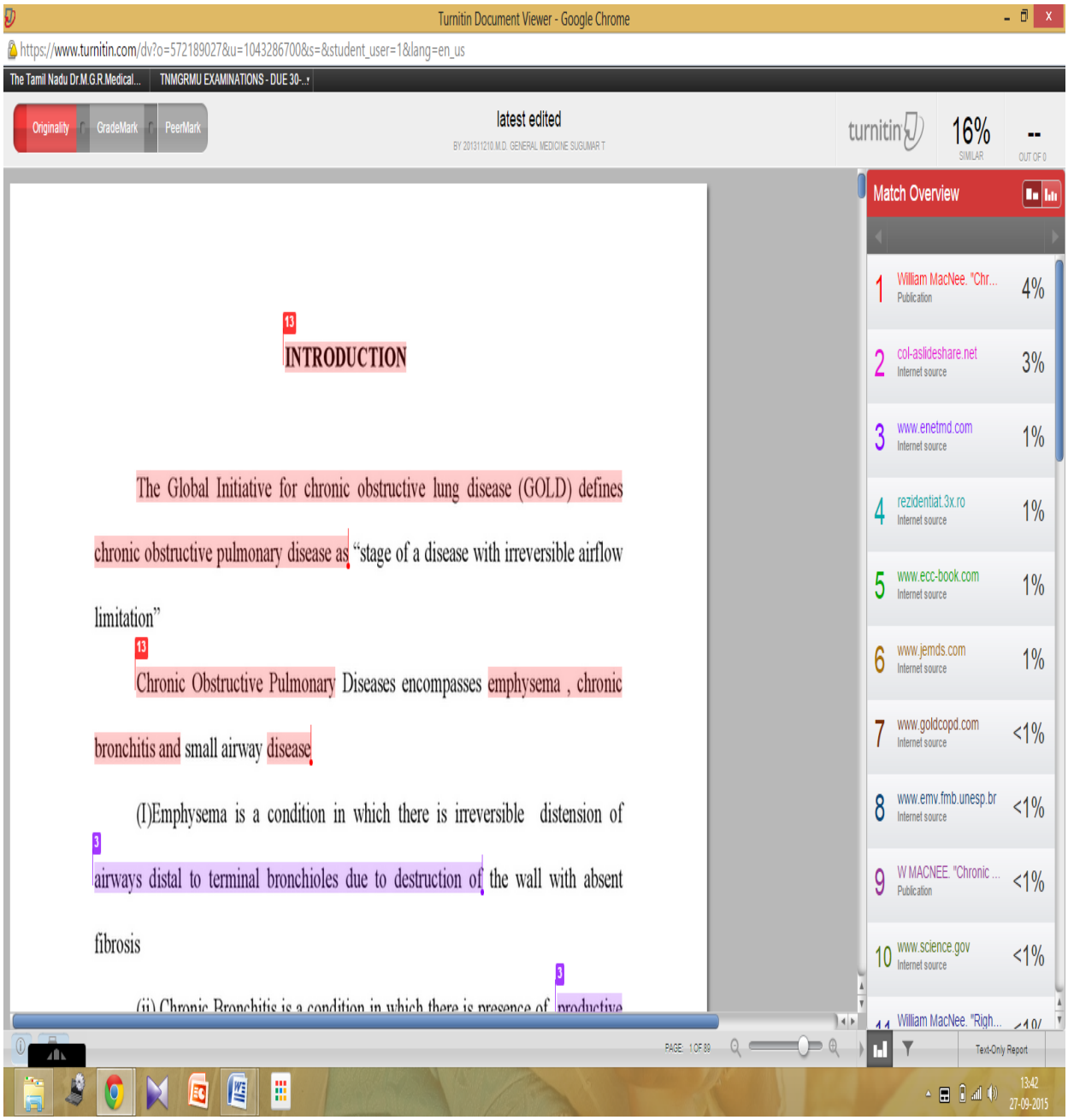
My sincere thanks to all the patients who cooperated for this study, without whom this study would have been impossible.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The Global Initiative for chronic obstructive lung disease (GOLD) defines chronic obstructive pulmonary disease as "stage of a disease with irreversible airflow limitation"
Chronic Obstructive Pulmonary Diseases encompasses emphysema , chronic bronchitis and small airway disease
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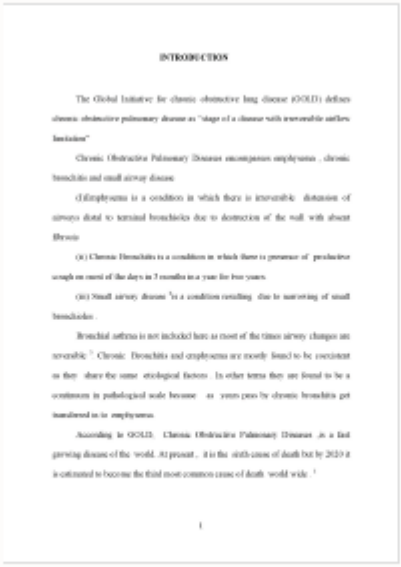

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(i)Emphysema is a condition in which there is irreversible distension of airways distal to terminal bronchioles due to destruction of the wall with absent fibrosis

(ii) Chronic Bronchitis is a condition in which there is presence of productive cough on most of the days in 3 months in a year for two years.

(iii) Small airway disease [3]is a condition resulting due to narrowing of small bronchioles .

Bronchial asthma is not included here as most of the times airway changes are reversible [3] Chronic Bronchitis and emphysema are mostly found to be coexistent as they share the same etiological factors . In other terms they are found to be a continuum in pathological scale because as years pass by chronic bronchitis get transformed in to emphysema

According to GOLD, Chronic Obstructive Pulmonary Diseases ,is a fast growing disease of the world. At present , it is the sixth cause of death but by 2020 it is estimated to become the third most common cause of death world wide . [1]

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.....A STUDY OF CLINICAL PROFILE OF COPD.....

.....CHRONIC OBSTRUCTIVE PULMONARY DISEASE.....

submitted by Dr. .....T. SUGUMAR..... **of**

Dept. of .....GENERAL MEDICINE..... **Thanjavur Medical College, Thanjavur**

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ABBREVIATIONS

COPD: Chronic Obstructive Pulmonary Disease

GOLD : Global Initiative for Chronic Obstructive Lung Disease

FVC: Forced Vital Capacity

FEV1: Forced Expiratory Volume in 1st sec

DLCO: Diffusing Capacity for Carbonmonoxide

ECG: Electrocardiogram

CT: Computed tomography

α1 AT: α1 Antitrypsin

LTOT: Long Term Oxygen Therapy

CONTENTS

A STUDY ON CHRONIC OBSTRUCTIVE PULMONARY DISEASES

AUTHOR: Prof. Dr.K.Nagarajan, **Dr.SUGUMAR T**

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BACKGROUND: In India , Chronic Obstructive Pulmonary Diseases rank second only after pulmonary tuberculosis , which is the leading disease affecting the respiratory system. The incidence of Chronic Obstructive Pulmonary Disease is high in the middle-aged subjects.This study documents the age and sex distribution of Chronic Obstructive Pulmonary Diseases and the exposure of risk factors of COPD in Thanjavur population. Further more this study is to elucidate the relationship of clinical symptom and spirometry abnormalities and also the incidence of right heart failure in Chronic Obstructive Pulmonary Diseases.

AIMS & OBJECTIVES: To study the age and sex distribution and the prevalence of various risk factors and to correlate the clinical symptoms with spirometry abnormalities in Chronic Obstructive Pulmonary Diseases. And to study the clinical incidence of Right Heart failure in Chronic Obstructive Pulmonary Diseases.

METHODS:

This study was conducted at Department of Medicine and Thoracic Medicine, Thanjavur Medical College during the period of Jan 2015 to

August 2015. Number of patients in this study is 60 cases. Those who complied with the inclusion and exclusion criteria were subjected to detailed history taking and clinical examination and necessary blood tests and imaging

RESULTS: COPD has male predominance as evidenced by 9:1 ratio of Male to Female due to high prevalence of smoking habits observed in males. Cigarette smoking was the major risk factor for COPD in this study. Cough / Cough with expectoration of sputum was the major clinical symptom observed in this study. Acting accessory muscles of respiration with pursed lip breathing was the major clinical sign observed in this study.

CONCLUSION: COPD is the disease of aged as majority of patients in the present study belong to the age group of 50 – 80 years. Spirometry is the mandatory investigation to diagnose and assess the severity of COPD. Most number of cases had severe airway obstruction which was not reversible. High flattened diaphragm and hyperlucent lungs were the most common chest x-ray finding observed in this study.

KEY WORDS

Spirometry, chronic obstructive lung disease, pack years

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According to GOLD, Chronic Obstructive Pulmonary Diseases ,is a fast growing disease of the world. At present , it is the sixth cause of death but by 2020 it is estimated to become the third most common cause of death world wide . [1]

In India , Chronic Obstructive Pulmonary Diseases rank second only after pulmonary tuberculosis , which is the leading disease affecting the respiratory system. [19]

The incidence of Chronic Obstructive Pulmonary Disease is high in the middle-aged subjects with males being the most commonly affected due to increased frequency of smoking among them . [22]

The disease prevalence is documented to be equal in both rural and urban regions . [19]

In this age of urbanisation and acculturation increase in smoking habits , increase in usage of gasoline automobiles and emergence of large scale industries leading to Air contamination has led to increase in incidence of Chronic Obstructive Pulmonary Diseases.

Chronic Obstructive Pulmonary Diseases has also been found to be linked with Low birth weight, malnutrition, recurrent respiratory infections in young age. [24]

Spirometry is the most robust test of airflow limitation in patients with Chronic Obstructive Pulmonary Diseases. [3]

A low FEV_1 (FEV_1<80%) with FEV_1 / FVC ratio < 0.7 and < 15% reversibility of airflow obstruction to bronchodialators is the diagnostic criteria for COPD. [2](FEV_1 – Forced Expiratory Volume in 1 sec. FVC – Forced Vital Capacity)

This study was conducted at Thanjavur Medical College during the period of January 2015 to June 2015. This study documents the age and sex distribution of Chronic Obstructive Pulmonary Diseases and the exposure of risk factors of COPD in Thanjavur population

Further more this study is to elucidate the relationship of clinical symptom and spirometry abnormalities and also the incidence of right heart failure in Chronic Obstructive Pulmonary Diseases.

AIM OF THE STUDY

1. To study the age and sex distribution in Chronic Obstructive Pulmonary Diseases.

2. To study the prevalence of various risk factors in Chronic Obstructive Pulmonary Diseases.

3. To correlate the clinical symptoms with spirometry abnormalities in Chronic Obstructive Pulmonary Diseases.

4. To study the clinical incidence of Right Heart failure in Chronic Obstructive Pulmonary Diseases.

REVIEW OF LITERATURE

Chronic obstructive pulmonary disease have been long studied from the times of Laennec . Though there has been many mechanical explanations of the disease, the association with smoking was not made untill the start of first half of the 20th century . But the association of heart failure with lung disease has been well established from earlier times.

The earliest documentation of imbalance of enzymes and Anti enzymes as risk factor of emphysema was made by OPIE , et al in 1905.[3]

Then there were various other research works on emphysema . One such was done by Kountz and Alexander in 1934 which concluded that heart was most commonly affected in emphysema .Another in 1950s by Liebow et al proposed a vascular atrophy model of the disease . In 1960 Gross and his associates observed the presence α_1 antitrypsin deficiency in emphysema patients leading to the development of proteinase and antiproteinase hypothesis in emphysema .

For the first time in year 1956 , the term chronic bronchitis was used by Medical research council in its journal for a condition with chronic cough with expectoration were other causes like pulmonary tuberculosis and bronchiectasis were excluded .

The relationship between smoking with chronic cough and sputum production was established by Higgins in year 1959. The resultant pathological changes in

airways, due to smoking was observed by Owen and Campell in late 1960s .

There has been numerous works on effects of chronic obstructive disease on cardiovascular system some of those significant studies are described below.

In 1971 Boushy SF et al., studied 108 patients of Chronic Obstructive Pulmonary Diseases. The ECG was correlated with pulmonary function Test and haemodynamic data.

In 1972 Benjamin Burrows et al, done a seven year study of 50 cases of Chronic Obstructive Pulmonary Diseases whose cardiac status had been evaluated by cardiac catheterization when they were stable, showed that their longevity was inversely related with pulmonary vascular resistance.

In 1973 Bougly and Colleagues published a series of papers on prognosis factors in Chronic Obstructive Pulmonary Diseases and prognosis values of lung function test in Chronic Obstructive Pulmonary Diseases.

In 1981 Matthay et al listed the electro cardio graphic changes characteristic of RV and RA enlargement in COPD and cor pulmonale.

In 1987 Danchin N et al., used 2D echocardiography to study right side cardiac parameter and RV hypertrophy and showed the correlation between Echocardiography changes and clinical symptoms.

In 1990 Migures M et al showed that usefulness of pulsed Doppler Echocardiography to detect pulmonary artery hypertension in patients with COPD.

EPIDEMIOLOGY

Chronic Obstructive Pulmonary Diseases is responsible for considerable morbidity and mortality in the community especially among the older people. This disease places a major burden on the resources of health care system because of their protracted course spanning over long period of time.

COPD is a preventable disease and thus is of considerable public health importance .

Males are the most commonly affected due to high prevalence of smoking among them compared to females.

Exacerbation of the COPD occurs more during winter season due to the increase in environmental trigger factors in that season .

PREVALENCE IN INDIA

Chronic Obstructive Pulmonary Diseases is the second most common lung disorder after pulmonary tuberculosis.

Incidence is higher in males due to higher prevalence of smoking rates among them . It is a disease of middle aged and elderly people and is less common below the age of 35 years.

Previous Studies conducted in other parts of the country, reported that the prevalence of chronic bronchitis was as high as 16% in people above 40 years of age . Due to extremes of climate in North India , they are found to have higher prevalence of COPD compared to South India .

Another study by Bhattacharya et al showed that the prevalence of chronic bronchitis in the age greater than 30 years was 57 / 1000 in a rural population . The study also documented male preponderance and the incidence of the disease directly proportional to the increasing age and number of pack years of smoking.

PREVALENCE IN WESTERN COUNTRIES:

In USA, it is estimated that there are around 16 million cases of COPD of whom about 14 million have chronic bronchitis and other 2 million have emphysema. The Male : Female ratio ranges from 4 – 6% / 1 – 3%. It is the third leading cause of death in U.S. with around 1 lakh per year .

In UK, a prospective study involving 40,000 medical practitioners, showed that death due to chronic bronchitis was higher in smokers and was directly proportional to the duration of smoking.

AETIOLOGY

Chronic Obstructive Pulmonary Diseases is characterized by areduced Forced Expiratory Volume in 1st second (FEV_1) and it progresses with accelerated rate of decline in FEV_1.The reduction in FEV_1 can occur by any of the three pathways.

1. In case of disturbed childhood growth and development, lower peak is attained in early adulthood followed by a normal rate of decline with ageing. This can be seen in case of early childhood infection and in case of exposure to passive smoking.
2. There will be a normal growth and development with premature peak in adulthood due to asthma or passive smoking and subsequent normal decline

.

3. There will be a normal attainment of growth and development and subsequent adulthood peak but followed by accelerated decline. This occurs due to active smoking and due to environmental exposures.

RISK FACTORS:

Smoking

Cigarette smoking is the principal cause of disease in about 90% of patients with COPD . There is a dose dependent response relationship between the number of pack-years of tobacco consumed and morbidity and mortality due to COPD. Pipe and cigar smokers are found to have reduced morbidity ratios compared to cigarettes, due to much lesser amount of inhaled toxicants with their use . There is still now no clear cut evidence between the usage of Electronic cigarettes and incidence COPD. According to the British Thoracic Society guidelines most of the COPD patients with have at least 20 pack years of smoking history. The highest annual rate of decline in FEV_1 in an average cigarette smoker is about 50ml, which is nearly double the average value of decline in nonsmokers.s

In nonsmokers, the decline in FEV_1 begins at mid 30s years which may occur earlier than this age in smokers. Stoppage of cigarette smoking decrease the rate of decline in FEV1 but it does not produce improvement in FEV_1.

Cigarette smoking has many harmful effects which includes the impairment of ciliary movements of respiratory epithelium, inhibition of alveolar macrophage function and the continuous irritant action leads to hyperplasia and hypertrophy of

mucus secreting glands lining the respiratory tract . The inhaled toxicants from Cigarette smoke also inhibits anti-proteases resulting in increased activity of proteolytic enzymes released from polymorphonuclear leukocytes . Smoking is also associated with increased airway responsiveness, this is linked with rapid progression of COPD in these patients . The earliest mechanical defect identified in a smoker is obstruction of small airways .

Air Pollution

Air pollution is the next other common risk factor linked with COPD. This association is best demonstrated by the increase in the incidence and mortality in COPD patients in heavily industrialized urban areas. The heavy pollution with sulfur dioxide and particulate matter is linked with increased episodes of exacerbations of bronchitis .

The traditional cooking fuels such as wood, cow dung cake, etc., along with poorly ventilated houses in most of under developed countries contributes to the development of chronic bronchitis.

Socio Economic Status

There is a strong association between socio economic status and respiratory symptoms , as occupation is linked with respiratory symptoms , this was demonstrated in a study in which twins were included as subjects by Hrubec et Al . There are many other studies which observes an inverse relationship between per capita income and the obstructive lung disease.

Occupation

Chronic bronchitis is more prevalent in workers who are engaged in occupation exposing them to inorganic or organic dusts, or to other noxious gases. There are surveys which have demonstrated an accelerated decline in lung function in workers for example who are employed at plastic plants and in those exposed to Toluene disouganate etc. Exposure to cadmium has been directly linked to the development of emphysema.

Recurrent Respiratory Infections

The occurence of acute respiratory illness are higher in frequency in patients with chronic bronchitis. The Epidemiological studies however have implicated , recurrent respiratory illness as one of the major factors associated with development and progression of chronic airway obstructive disease .

Airway hyper responsiveness and atopy:

Though airway hyper responsiveness is a definitive feature of asthma , it is also seen in many COPD patients but with less than 15% of reversibility of obstruction by usage of bronchodilators.

Growth And Nutrition

There are evident Studies that show malnutrition accelerates decline in ventilatory function. There is also evidence that severe viral pneumonia early in life may lead to chronic obstruction of small airways.

GENETIC FACTORS:

α_1, Antitrypsin Deficiency

α_1 AntiTrypsin (α_1AT) is a polymorphic glycoprotein which is responsible for the majority of anti-protease activity in the serum. The genetic material of this protein is mapped on chromosome 14q 32 . The most commonest alleles resulting in deficiency is termed as ZZ (Cor Pi^{ZZ}Phenotype) results from a singleamino acid substitution Glu → Lysin the position 342 , which causes spontaneous polymerization of the polypeptide, leading to impairment in release of the protein into circulation from the liver.

It is most common in people of European descent with incidence 1:2000 compared to 1:7000 in the people of African and Asian lineage. α_1AT deficiency accounts for only 2% of observed cases of emphysema. These patients present with early development of emphysema, chronic bronchitis or bronchiectasis. They present usually in early forth decade of life with cough and dyspnea. Nearly 80% of this population have family history of lung disease with autosomal recessive inheritance. Pathologically the panacinar emphysema predominates and radiographic changes are more common in lower lobes.

Tobacco smoking is an extremely important cofactor for development of disease in α_1 AT deficiency. The average decline of FEV_1 is 100-130ml / yr for smokers and 50 to80ml / yr for ex-smokers of lifetime non-smokers. They are also at increased risk of developing hepatic cirrhosis.

PATHOLOGY

The pathologic changes of COPD involves large and small airways and the terminal respiratory unit.

-Hypertrophy of mucus producing glands.

-goblet cell hyperplasia

-reduced and sluggish movements of cilia

-mucosal edema

-hypertrophy of smooth muscles of bronchus

-reduced calibre of air spaces

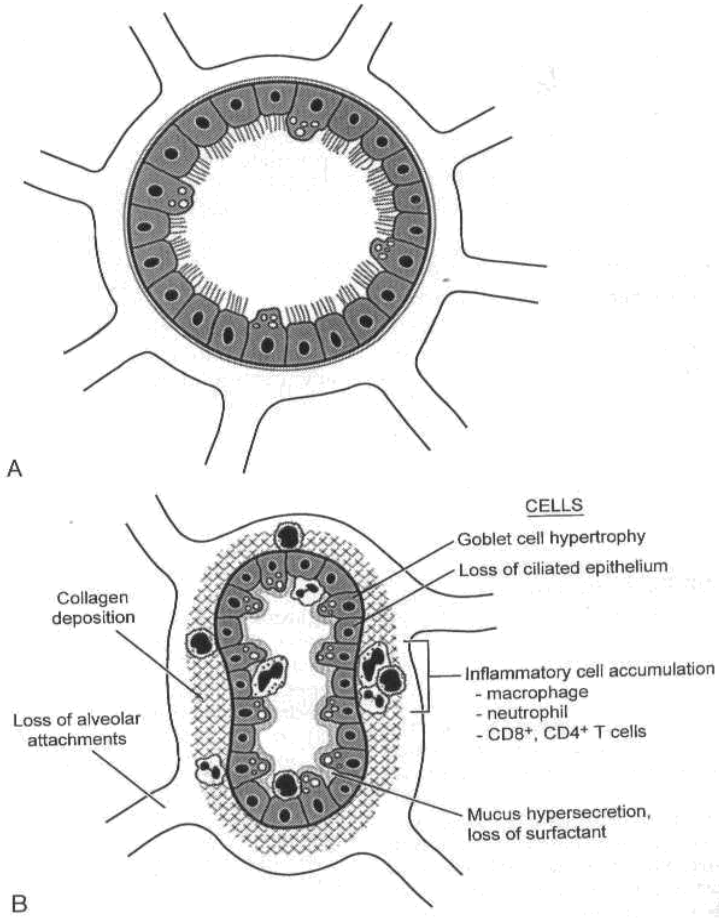


Figure 2. Mechanisms of small airway obstruction in chronic obstructive pulmonary disease (COPD). In contrast to a normal small airway (< 2 mm) *(A)*, note changes in small airways in COPD *(B)*. There are cellular changes including goblet cell hypertrophy, loss of ciliated epithelial cells, and accumulation of inflammatory cells. Mucus hypersecretion and loss of surfactant may also predispose to airway narrowing. Extracellular matrix remodeling is complex with both collagen deposition causing airway obstruction and loss of elastic fibers and hence alveolar attachments that tether open the airways.

mechanisms of airway obstruction in COPD

Pathogenesis of chronic bronchitis

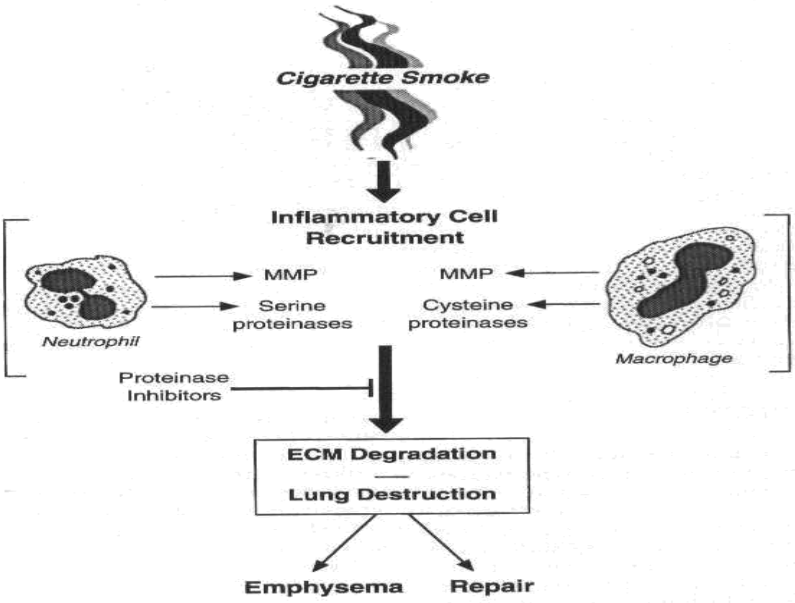


Figure 1. Pathogenesis of emphysema. On long-term exposure to cigarette smoke, inflammatory cells are recruited to the lung, they release proteinases in excess of inhibitors, and if repair is abnormal, this leads to airspace destruction and enlargement or emphysema. MMP = matrix metalloproteinase; ECM = extracellular matrix.

The major site of airflow limitation is seen in small airways . The narrowing of airways are due to goblet cell hyperplasia, infiltration of mucosal and submucosal layer by inflammatory cells, oedema, reactionary peribronchial fibrosis, intraluminal mucus plugs and increase in smooth muscle density.

In the large cartilaginous airways, chronic bronchitis is associated with hypertrophy of submucosal mucus producing glands. These anatomic changes can be assessed by Reid index which is a ratio of submucosal glands thickness to that of bronchial wall. In case of chronic bronchitis patients it is 0.52 ± 0.08otherwise normally it's around 0.44± 0.09.

Pathogenesis of emphysema

The initial step in development of Emphysema is the increase in the number and size of alveolar fenestrate which eventually results in destruction of alveolar septae and their attachments to terminal respiratory bronchioles are lost .In centriacinar emphysema the distension and destruction are mainly limited to the respiratory bronchioles with relatively less changes peripherally in the acinus. Panacinar emphysema involves both central and peripheral portions of acinus.

Chronic exposure to cigarette smokes, fumes and dust leads to the recruitment of inflammatory cells in to the terminal air spaces of the lung. These cells then release elastolytic proteinases that damage the extra cellular matrix of the lung.

The ineffective control of destruction of lung parenchyma result in emphysema.

PATHOPHYSIOLOGY

Airflow limitation:

Airflow limitation and increased airway resistance is caused by loss of elastic recoiling of lung during passive exhalation due to destruction of alveolar walls , or due to increased collapsibility of small airways through loss of radial traction on airways, or to increased resistance due to intrinsic narrowing of small airways.

Hyperinflation:

The residual volume and the functional residual capacity (FRC) are higher than normal. In addition prolongation of expiration is associated with obstruction ,

leads to dynamic increase in Functional residual capacity (dynamic hyperinflation).Dynamic hyperinflation in turn contributes to discomfort associated with air flow obstruction by flattening of diaphragm .

Impaired Gas Exchange:

Ventilation and perfusion mismatching is always present. When the mismatching is severe, impairment of gas exchange is reflected as decreased PaO2 in the arterial blood gas analysis. The mismatch is due to Small airway narrowing leading to decrease in ventilation of their distal alveolar acini were the alveolar capillaries remain intact .

Pulmonary Circulation in COPD

Pulmonary arterial hypertension develops late in the course of COPD due to the development of hypoxia and usually hypercapnia. It is the major cardiovascular complication of COPD and is associated with the development of right ventricular hypertrophy (cor pulmonale) . It has a poor prognosis.

Factors contributing to pulmonary artery hypertension in COPD

(i) ABNORMAL BLOOD GAS TENSIONS

Hypoxia

In COPD there is a negative correlation between oxygen saturation of the blood and pulmonary artery pressure. Hypoxemia is known to be a potent pulmonary arteriolar constrictor .As the disease progresses in severity , there is more arterial desaturation which results in increase in pulmonary artery pressure. The acute

worsening of pulmonary hypertension is linked with COPD exacerbation. Pulmonary artery pressure (Ppa) can also increase acutely during rapid eye movement of sleep which is associated with hypoxia , and it has been suggested that recurrent nocturnal pulmonary hypertension can result in pathologic changes in pulmonary vessels and fixed hypertension.

Hypercapnea:

In patients with COPD there is a positive correlation between arterial CO_2 pressure ($PaCO_2$) and pulmonary artery pressure. The mechanism could be a change in lung mechanics due to hyperventilation induced by hypercapnea or the potentiation of hypoxic pulmonary vascoconstriction.

Acidemia:

Hypoxia and acidemia act synergistically to produce pulmonary vasoconstriction in patients with COPD. Thus for a given oxygen saturation, the mean Ppa is higher with associated increase in arterial hydrogen ion concentration.

(ii) EFFECTS OF ABNORMAL PULMONARY MECHANICS

Changes in airway resistance may augment pulmonary vascular resistance and increase in pulmonary artery pressure, correlating with decrease in FEV_1.

(iii) EFFECTS OF INCREASED CARDIAC OUTPUT

In patients with COPD (in whom the vascular bed may be reduced) even small

increase in flow that occurs during exercise may increase pulmonary artery pressure.

(iv) EFFECTS OF BLOOD VISCOSITY

Polycythemia can develop secondary to chronic hypoxia in COPD patients, this contributes to blood viscosity which also adds up to the pulmonary arterial hypertension.

(v) ROLE OF PULMONARY ENDOTHELIUM

Endothelial dysfunction causes reduced nitric oxide synthesis which leads to hypoxia resulting in development of pulmonary artery hypertension. .

Thus the role of nitric oxide which prevents the increase in pulmonary vascular resistance is lost in this disease. Nitric oxide also inhibits cell proliferation in the pulmonary vessel walls and hence prevents the vascular remodeling. Circulating levels of endothelin has found to be increased in patients with COPD and pulmonary hypertension.

PATHOLOGY

The changes in pulmonary circulation occur characteristically in the pulmonary arteries in patients with COPD. In the patients with COPD characteristic changes .An early change is the increased intimal thickness in small pulmonary arteries that occur due to accumulation of smooth muscles that are laid down longitudinally along the length of the vessel. The medial hypertrophy in muscular pulmonary vessels, has also been reported in patients of COPD who develop sustained pulmonary arterial

hypertension. Pulmonary thromboses may also occur in patients with COPD which may be secondary to peripheral airway inflammation.

These structural changes rather than simply hypoxia ,vasoconstriction is the major factor in the development of sustained pulmonary hypertension in patients with COPD.

PULMONARY HAEMODYNAMICS COPD

Pulmonary hemodynamics depends on the severity of the disease . in mild cases , pulmonary artery pressure is normal iat rest and is moderately elevated during exercise .Cardiac parameters such as output , systolic and diastolic pressures are also normal.

In case increase in severity associated with chronic hypoxia and hypercapnia , pulmonary artery pressure shoots up even at rest. However in cases of clinically stable severe COPD pulmonary artery pressures are only moderately elevated .

CONSEQUENCES OF PULMONARY HYPERTENSION IN COPD

Chronic bronchitis and emphysema usually coexist pathologically. Those patients with either predominant chronic bronchitis or emphysema from a minority at the either ends of the disease spectrum of COPD.

The blue and bloated type also know as type B or non fighter was though to characterize the bronchial type of disease.

These patients have hypoxemia, hypercapnea and secondary polycythemia, leading to early development of pulmonary hypertension .. In these cases there occur frequent repeated episodes in right heart failure associated with right ventricular hypertrophy

In contrast the pink and puffing variety also known as type A or fighter, were though to represent the emphysematous patients characterized by severe breathlessness, but with preservation of blood gas values and thus no pulmonary hypertension, at least until the later stages of disease.

It was now known that the degree of mucous gland hypertrophy indicative of chronic bronchitis was similar whatsoever the clinical pattern and that more than 50% of patients with blue and bloated clinical pattern had severe emphysema.

THE NATURAL HISTORY OF UNTREATED PULMONARY HYPERTENSION IN COPD

The progression of pulmonary hypertension in COPD is slow. The elevation in pulmonary artery pressure is small and it rarely reaches the levels of primary pulmonary hypertension. Weitzenblum and colleagues found a change of 3mm Hg in the pulmonary artery pressure per year and 33% of patients showed a rise greater than 5mm Hg over 5 years who had worsening of hypoxemia and hypercapnia

Inspite of the slow progression the presence of pulmonary artery hypertension implies a poor prognosis.

One study shows that the 4 years survival rate was 40% in patients with pulmonary hypertension compared to 72% in patients whose pulmonary artery

pressure was normal (less than 20mm Hg).

The other variable which correlates significantly with survival are PaO_2, $PaCO_2$, FEV_1 and the presence of peripheral oedema.

The pulmonary hypertension reflects the severity of the disease and predicts the prognosis of COPD.

COR PULMONALE

Cor pulmonale is defined as right ventricular hypertrophy and dilatation secondary to pulmonary hypertension caused by disease of the lung parenchyma and / or pulmonary vasculature, unrelated to both sides of heart.

In an European study , enrolling large population showed that incidence of right ventricular failure increases as air flow limitation worsens in COPD patients, that is 40% of patients had right ventricular failure when $FEV_1 < 1.0L$ and 70% had failure when $FEV_1 < 0.6L$. The prevalence of cor pulmonale is also higher in patients with hypercapnia , hypoxemia and polycthemia.

In clinically stable COPD patients ,right ventricular function is near normal even with increased pulmonary artery pressure . But in clinically unstable patients in respiratory failure with failing right ventricular function the pulmonary artery pressure is very much increased compared to stable.

There is increasing evidence that the oedema which develops late in the course of the disease in patients with COPD may not be entirely due to right ventricular failure. The fluid and electrolyte imbalance in COPD is due to the presence of associated hypoxemia and hypercapnia. There is decrease in renal blood flow in

patients with hypoxic COPD due to the increase in release of catecholamines associated with hypercapnia. Arginine vasopressin levels are high in patients with COPD compared to normal population. There is also evidence of activiation of rennin-angiotensin-aldosterone axis. Thus peripheral oedema. is a resultant of change in pulmonary hemodynamics interaction with fluid and electrolyte imbalance.

CLINICAL FEATURES

Symptoms

The most common clinical presentation of COPD is breathlessness on exertion mostly accompanied with wheeze and cough with thick productive sputum .Most patients have a smoking history of minimum 20 pack years before the presentation . The mean age is around fifth decade.

Breathlessness is the most debilitating of all symptoms ,which is due to loss of lung function over time which is first noted on climbing or exertion. The presence of breathlessness indicates the extent of impairment of airway function. By the time when patient seeks the medical help FEV_1 has fallen significantly . When FEV_1 has fallen to 30% or less breathlessness is generally present even on minimal exertion. In most patients with COPD cough is the first symptom to appear and is the neglected one, leading to time lag for medical approach. Sputum is usually mucoid and thick in character, but becoming purulent during exacerbations, which is linked with bacterial infections. Though there is copious secretions only small amount of sputum less than 60ml per day is expectorated due to narrowing of airway and increased viscosity of secretions .

As the disease progresses the frequency and severity of exacerbations increases .

Wheezing is present but it is not specific and it does not indicate the severity of the obstruction .

Weight loss and anorexia are seen in severe COPD.

Physical Signs

These is no specific signs for the disease and expect that the signs depend on the degree of air flow limitation and over inflation. In early stage of the disease the only abnormal findings is wheeze on forced expiration. There can be inspiratory and expiratory coarse crackles associated with mucus production and cough . In case of forced expiratory time longer than 6 seconds in more advanced disease, the breathing pattern is characteristic with a prolonged expiratory phase. Some patients adopt pursed-lip breathing on expiration which reduces expiratory airway collapse and improves oxygenation . The use of accessory muscles of respiration like alae nasi , sternocleidomastoid is seen in advanced disease. They may also adopt the tripod position by leaning forward, supporting themselves with their arms to fix the shoulder girdle and allowing the use of pectoralis and latissimus dorsi to augment chest wall movements.

In later stages of the disease the chest wall becomes barrel shaped with increase in anterior posterior diameter, horizontal ribs, prominence of the sternal angle and wide subcostal angle . An inspiratory tracheal tug may be detected.

The diaphragm also assumes a horizontal position which helps to pull the lower ribs during inspiration (Hoover's sign).

On percussion there is reduced cardiac and hepatic dullness, due to over inflation of lungs

Breath sounds may have a prolonged expiratory phase or can be diminished uniformly .

COMPLICATIONS OF COPD

Acute mucopurulent exacerbations with secondary infections

Respiratory failure

Pulmonary bullae formation

Bullae rupture

Pneumothorax

Cor pulmonale

RADIOLOGY

Chest X-ray:

There are no specific findings for chronic bronchitis in plain chest x Ray . Bronchial wall thickening may be noted as parallel line opacities.

The following Radiographic features are seen in emphysema:

- Share-sheath treeches.
- Low flattened diaphragm: The border of the diaphragm in the midclavicular line below the seventh rib.
- Height of patients lung being greater than 29.9 cm.
- An obtuse costophrenic angle.
- Reduction in size and number of pulmonary vessels particularly in periphery of lung.
- Heart shadow is vertical and narrowed .
- In lateral film , there can be increase in the retrosternal airspace.

Computed tomography:

The high resolution computed tomography has greater sensitivity and specificity than plain chest X-ray for emphysema but it is rarely required except for diagnosis of bronchiectasis and to rule out bullous lung disease.

SPIROMETRY

Because of imprecisions in the clinical findings, objective evaluation for detecting the presence, assessing severity and reversibility of airflow obstruction is essential for the diagnostic evaluation of COPD.

Spirometry is the most robust test of airflow limitation in patients with COPD.

Forced expiratory volume in one second (FEV_1) is recommended as the measurement of choice as

- FEV_1 is a reliable objective measurement.
- It is simple and relatively quick to measure in all stages of the disease.
- The forced expiratory manoeuvre records $FEV_1$and also FVC . FEV_1 / FVC ratio less than 70% is diagnostic of airway obstruction.
- FEV_1 predicts future morbidity and mortality.
- Serial measurement provides evidence of disease progression.

Global initiative for chronic obstructive lung disease (GOLD) group has reclassified COPD in June 2003 based on spirometery values.

FLOW VOLUME LOOPS

Expiratory flow at 75% or 50% of vital capacity have been used as a measure of airflow limitation and provide complementary information to the usual volume time plot. There are problems with the reproducibility of these measurements and hence not preferred for routine clinical use.

Reversibility to bronchodilators

Reversibility tests are important because

1. It distinguishes those patients with marked reversibility (at least 12% or 200ml of FEV_1) who have underlying asthma.

2. It aids in future management.

3. The FEV_1 after bronchodilator is the best predictor of survival.

It is usually recommended that the response to bronchodilators be assessed either using repeated doses from metered dose inhaler or via the nebulised route.

DIFFUSING LIMIT OF LUNG CARBON MONOXIDE (DLCO)

DLCO values are below normal in many patients with COPD .There is a relationship between gas transfer and microscopic emphysema but the severity of emphysema in an individual patient cannot be predicted from this.

ARTERIAL BLOOD GAS ANALYSIS

Measurement of arterial blood gas is essential in patients with COPD especially in exacerbations to assess the degree of hypoxaemia and hypercapnia .

OTHER TESTS

Patients with pulmonary hypertension and cor pulmonale with normal day time blood gases should be evaluated for nocturnal desaturation by overnight oximetry.

α_1- AT level are not routinely needed but should be obtained incase of occurrence of COPD in non smokers, and in those associated with bronchiectasis, cirrhosis without apparent risks, premature emphysema in patients under 40 years with unremitting disease and in individuals with family history of AT deficiency.

METHOD OF ASSESSING CARDIAC FUNCTION IN PATIENTS WITH COPD

The pulmonary haemodynamics and right ventricular function an be assessed by measuring pressure and flow which involves the use of invasive techniques like cardiac catheterizaiton.

The non invasive techniques which include radiography ,electrocardiography, echocardiography, radionucleide ventriculography and magnetic resonance imaging have proved to be useful in assessing COPD patients .

CLINICAL ASSESSMENT

Clinical examination is relatively insensitive in detecting pulmonary hypertension or right ventricular dysfunction in patients with COPD, as clinical signs are often obscured by the hyperinflation of the lungs .

Physical signs that indicate the presence of pulmonary hypertension are loud pulmonary component of S_2 with early systolicclick,and an early diastolic murmur in case of pulmonary regurgitation .

Jugular venous pressure is difficult to assess in these patients due to large swings in the intrathoracic pressure.

Peripheral oedema can be present due to other related causes such as hypoalbuminemia .

A left parasternal heave indicates right ventricular hypertrophy. The extra heart sounds and murmur of tricuspid regurgitation suggests right ventricular dysfunction, but these are again modified by hyperinflation.

CHEST X-RAY

The pulmonary artery width≥ 20mm relates directly to the presence of pulmonary arterial hypertension .The high value for hilar cardiothoracic ratio has 95% sensitivity and 100% specificity for the presence of pulmonary hypertension in patients with COPD.

These are used as initial screening test for the presence of pulmonary hypertension, but they do not predict the level of pulmonary artery pressure in individual patients.

ELECTROCARDIOGRAPHY

The voluminous lungs have an insulating effect and thus diminishes transmission of electrical potentials to the electrodes. The heart descends to a lower position in the thorax due to lowering of diaphragm and alters the position of heart relative to the conventional electrodes.

The electrocardiography abnormalities in COPD and with right heart involvement are [10]

- Decreased magnitude of electrocardiograph wave deflections.
- P-waves with right atrial enlargement, p-pulmonale i.e., tall peaked P waves in II, III and aVF (P wave > 2.5mm)
- QRS abnormalities: Right axis deviation and QRS > 90*. At times with extreme northwest QRS axis there is the $S_1S_2S_3$ syndrome. In precordial leads there is a general loss of R wave amplitude in all precordial leads. With right ventricular hypertrophy R/S amplitude in V_6<V1.

- Tendency for incomplete right bundle branch block.

Electrocardiography appears to be specific but has a low sensitivity in picking up right ventricular hypertrophy.

ECHOCARDIOGRAPHY

Because of the deficiencies of clinical examination in detecting pulmonary artery hypertension in patients who have COPD and because pulmonary artery pressure (Ppa) is a good predictor of prognosis in these individuals, a number of attempts have been made to develop noninvasive methods to estimate it. Echocardiographic measurements of systolic , diastolic and pulmonary pressures have been shown to correlate with Ppa measured by the cardiac catheterizaiton studies. [11]

The most useful and accurate method of estimating pulmonary artery pressure in patients with chronic obstructive pulmonary disease is systolic transtricuspid gradient calculated from tricuspid regurgitation detected by continuous wave Doppler echocardiography. Continuous wave Doppler determination of tricuspid regurgitation jet velocity and

application of modified Bernoulli's equation ($TG = 4V^2$, in which V is the velocity of tricuspid regurgitation jet and TG is the systolic right ventricular to right atrial pressure gradient across the tricuspid valve) permits reliable estimation of pulmonary artery pressure .

Two dimensional echocardiography can be used to assess right ventricular dimensions and wall thickenings and hence to detect right ventricular volume and pressure over load in patients with COPD.

Echocardiography can again be used to assess progression of disease or response to treatment by serial measurements of pulmonary artery pressure and right heart parameters.

MANAGEMENT OF COPD

I. Non Pharmacological measures.

II. Pharmacological measures.

III Ventilatory support

I. Non Pharmacological Measures

Health education: it can play a role in improving stills, ability to cope with illness.

i. Smoking cessation.

ii. Basic information about COPD.

iii. Self management skills / pulmonary rehabilitation

II. Pharmacological Measures

i. Nicotine replacement therapy in the form of gum, transdermal patch or inhaler is helpful in quitting smoking.

ii. The use of bupropion, a noradrenergic antidepressant, is associated with better abstinence rates.

iii. Bronchodilators are the main stay in the management of COPD.

iv. Inhaled therapy is the preferred route

v. Choice between β_2 agonist, anticholinergics, theophylline or combination therapy depends on availability and individual response in terms of symptom relief and side effects.

vi. Bronchodilators are prescribed on as needed, or on a regular basis to prevent or reduce symptoms depending on stage.

vii. Long acting bronchodilators are more effective and convenient than the short acting ones .

viii. Combining bronchodilators of different groups can improve efficacy and decrease the risk of side effects compared to increasing the dose of a single bronchodilator.

ix. Roflumilast phosphodiesterase 4 inhibitor has found to reduce the frequency of exacerbations.

VENTILATORY SUPPORT

Indications of ventilatory support in COPD

Severe respiratory failure

Respiratory rate >35

hypercarbia ($PaCO_2$ >60 mmHg)

Acidosis (pH <7.25)

Respiratory arrest

Altered mental status

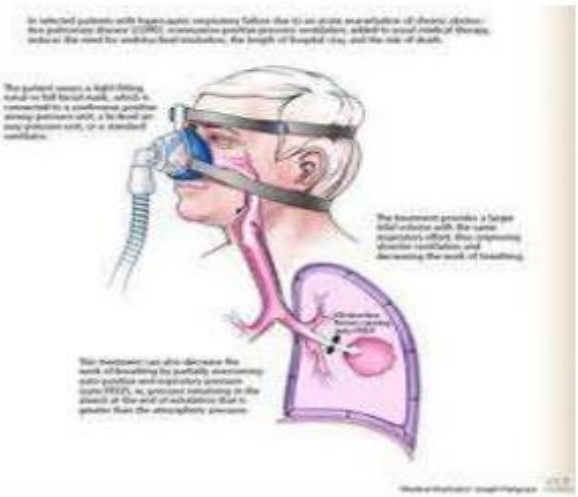
Hypotension,cardiac failure,shock

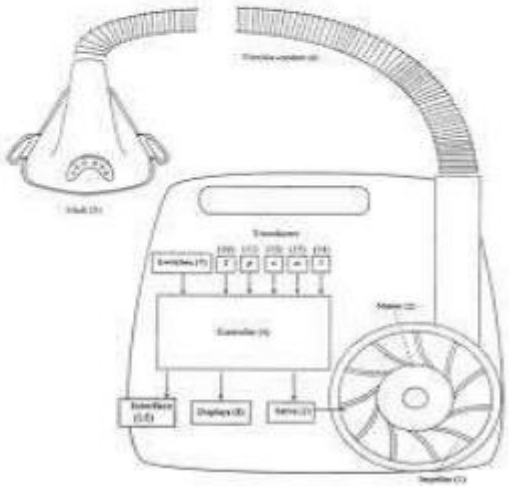
Generally for COPD patients Non Invasive ventilation is prefered over invasive ventilation due to difficult to wean from mechanical ventilation

NON-INVASIVE VENTILATORY SUPPORT IN COPD

NON INVASIVE POSITIVE PRESSURE VENTILATION

- During NIPPV, air enters the nose, mouth or both through the interface, which in turn is connected, to Positive Pressure Ventilator.





INVASIVE VENTILATION IN COPD

GLUCOCORTICOSTEROIDS

Inhaled glucocorticoids are appropriate for symptomatic COPD patients with an FEV_1< 50% predicted as it decreases the frequency of exacerbation .

But at present there is controversial reports on its effect over mortality reduction .

Present guidelines recommend a trial of 6 weeks to 3 months with inhaled steroids in patients with exacerbations more than two per year , to identify patients who may benefit from long term inhalation steroid therapy.

Long term treatment with oral steroids is not recommended in COPD because of its unfavourable risk ratio and Some of the side effects seen with long term steroid therapy are as follows

osteoporosis,

weight gain

,cataracts

,glucose intolerance and

increased incidence of infection

In case of acute exacerbations a 14 day course of 30 mg oral prednisolone is recommended by GOLD guidelines

Hyperglycemia is common complication reported in patients receiving short course glucocorticoid therapy

HOME OXYGEN THERAPY (HOT)

Long term oxygen administration (> 15 hours per day) to patients with chronic respiratory failure has shown increased survival rates in these patients .

In Nocturnal oxygen treatment trial , it has shown that there is significant reduction in pulmonary arterial pressure by use of either continous or nocturnal oxygen therapy for 6 months .

This study also showed that survival rate over next 8 years correlated with decrease in mean pulmonary arterial pressure during the first 6 months of treatment.

Long term oxygen therapy is indicated in Resting hypoxia were

- PaO_2 at or below 55 mmHg or SaO_2 at or below 88% with or without hypercapnia or
- PaO_2 between 55 mmHg and 60 mmHg or 90 % if there is evidence of pulmonary hypertension, peripheral oedema suggesting congestive heart failure, or polycythemia (Haematocrit > 55%).

Home oxygen therapy can be supplied in the form of compressed gas cylinders or oxygen concentrators. Oxygen concentrators are most convenient and cost effective.

OTHER PHARAMACOLOGIC TREATMENT

α_1 ANTITRYPSIN AUGMENTATION THERAPY:

Young patients with severe α_1 AT deficiency (< 11mic. moles) and established emphysema may be candidates for the same.

ANTIBIOTICS:

Bacterial infection is found to be major precipitant of acute exacerbations . Recently azithromycin because of its antibiotic and anti inflammatory property has found to reduce exacerbation of COPD by administering daily dose.

They play major role in the treatment of exacerbations

VACCINES:

Influenza vaccines annually can reduce serious illness and death in about 50% of those affected . Polyvalent pneumococcal vaccine is also has been recommended .

Surgical interventions

Lung volume reduction surgery Lung transplantation

MATERIALS AND METHODS

This study was conducted at Department of Medicine and Thoracic Medicine, Thanjavur Medical College during the period of Jan 2015 to August 2015.

TOTAL NUMBER OF PATIENTS IN THIS STUDY: -

Number of patients in this study is 60 cases.

INCLUSION CRITERIA: -

The cases in this study have following characters:

(i) Cases between the age group of 30 – 80 years of both sexes.

(ii) These cases having the symptoms suggestive of chronic airway obstruction like cough, cough with expectoration of sputum of more than 2 years duration, dyspnoea, and with (or) without swelling of both legs.

(iii) Cases in whom clinical diagnosis of COPD was made.

(iv) All the cases were subjected to spirometry and the presence of COPD was confirmed by post bronchodilator spirometry values of

i. $FEV_1 < 80\%$.

ii. $FEV_1 / FVC < 0.7$.

iii. Reversibility of obstruction < 15%.

(FEV_1 – Forced Expiratory Volume in 1 sec. FVC – ForcedVitalCapacity)

EXCLUSION CRITERIA: -

Case with history of the following diseases were excluded;

(i) Bronchial Asthma. (ii) Pulmonary Tuberculosis.

(iv) Suppurative lung disease (iv) Systemic Hypertension.

(v) CAHD. (vi) Primary Pulmonary Hypertension.

(vii) Sleep Apnoea syndrome (viii) Valvular Heart disease.

PROCEDURE:

With above inclusion and exclusion criteria a proforma wasprepared to meet the objectives of the study.

GEOGRAPHIC DISTRIBUTION:

Patients were from Thanjavur Town, Pattukottai, and rural areasof Thanjavur, Pattukottai and Ariyalur districts.

All the patients were subjected as follows;

1) Detailed History.

2) Smoking History. i) Age at which smoking was started.

ii) Pack – years was calculated by formula.

$$\text{Pack Years} = \frac{\text{no of cigarrates smoked /day}}{20} \times \text{No. of years of smoking}$$

3) General examination and examination of Respiratory system, and other systems.

4) Examination specifically for signs of right heart failure like raised JVP, congestive hepatomegaly and pedal edema.

5) Conventional 12 lead Electro cardiography was taken for ECG changes of COPD and Right Heart failure.

6) X-ray chest PA view and left lateral view.

7) Spirometry: - Spirometry was performed when the patient was clinically stable.

Test was performed with the patient comfortably seated, with clothes loosened. The patients were instructed to take a deep inspiration then close the lips around the mouth piece and blow out as hard and fast as possible, following deep inspiration.

Volume was obtained on the vertical axis of recording paper and time on the horizontal axis. The curve which was obtained is referred to as forced vital capacity curve.

Forced Vital Capacity (FVC) is the volume of air that can be forciblyexhaled (as fast as possible) after a maximal inspiration. It is expressed in litres.

Forced Expiratory Volume in one second (FEV_1)

It is defined as the volume of air expelled in the first second, from the start of maximum expiratory effort of the forced vital capacity. It is expressed in litres or percentage of predicted.

Forced expiratory volume in one second as a percentage of forced vital capacity (FEV_1 / FVC)

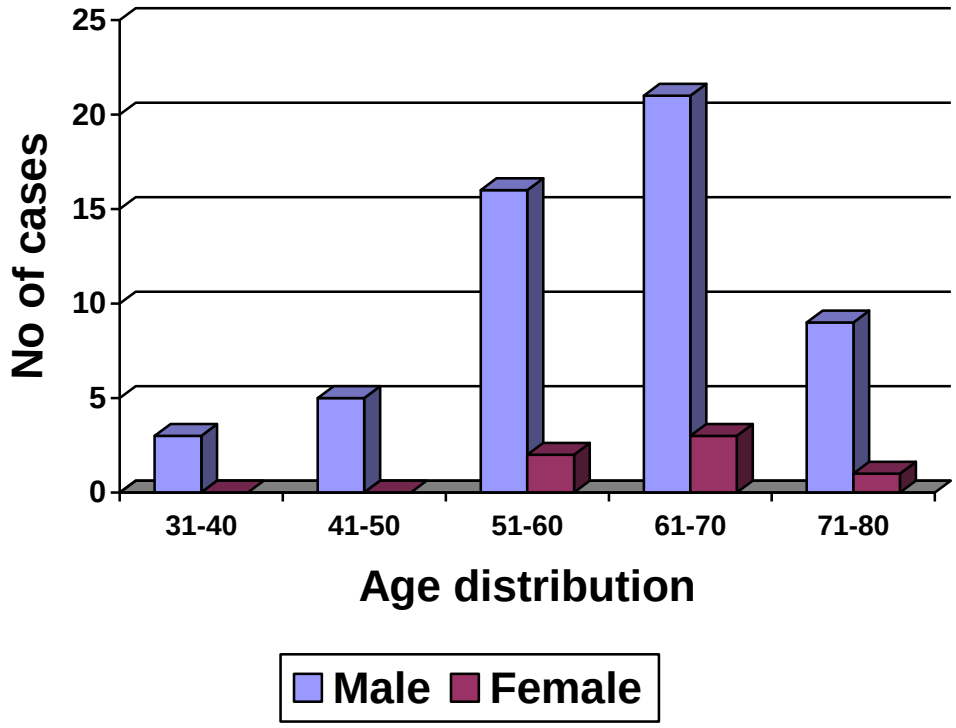
It is the percentage of forced vital capacity which is expelled in the first one second of maximal expiratory effort.

By using spirometry results patients were classified based on GOLD staging of COPD. Severity of clinical symptoms were correlated with GOLD staging of COPD.

Pack Years of smoking was compared with severity of COPD. Patients who showed clinical signs of right heart failure were subjected to Echo cardiography for confirmation. In echocardiography Mean maximum TR velocity was recorded in mt/sec and inserted in to the modified Bernoulli's equation ($4v^2$), thus calculating the trans tricuspid pressure gradient (TTPG) and the pulmonary artery pressure.

RESULTS AND OBSERVATION

BAR DIAGRAM SHOWING AGE DISTRIBUTION OF THE CASES



The study population had predominantly males in the age group of 50-70 years of age.

Table – 1

The following table shows the distribution in relation to the sex and percentage of prevalence among them .

Age In Years	**Male**	**Percentage**	**Female**	**Percentage**	**Total**	**Percentage**
31-40	3	5.5%	-	0%	3	5%
41-50	5	9.2%	-	0%	5	8.3%
51-60	16	29.6%	2	33.3%	18	30%
61-70	21	38.8%	3	50%	24	40%
71-80	9	16.6%	1	16.6%	10	16.6%
Total	**54**	**100**	**6**	**100**	**60**	**100**

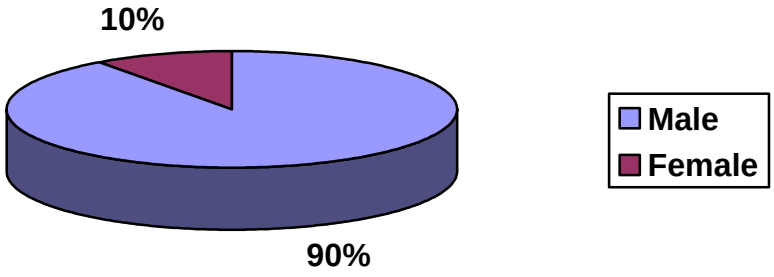
From the above table it is observed that the majority of cases among males were between 61 – 70 years of age constituting 38.8% and the minimum number of cases were in the age group of 31 – 40 constituting 5.5%.

The results collected from this study was tabulated into different variates and incidence of each variate was calculated in percentage.

Among females the majority of cases were in the age group of 61– 70 years constituting 40% and nil cases were obs erved in the age group of 31 – 50 years.

Both sexes put together the maximum cases were in the age group of 61 – 70 years constituting 40% of total cases an d minimum cases were observed in the age group of 31 – 40 years which constituted 5% of total cases.

PIE DIAGRAM SHOWING SEX DISTRIBUTION OF THE CASES



This diagram shows sex distribution of cases.A male preponderance was seen.

Sex distribution:

Table – 2

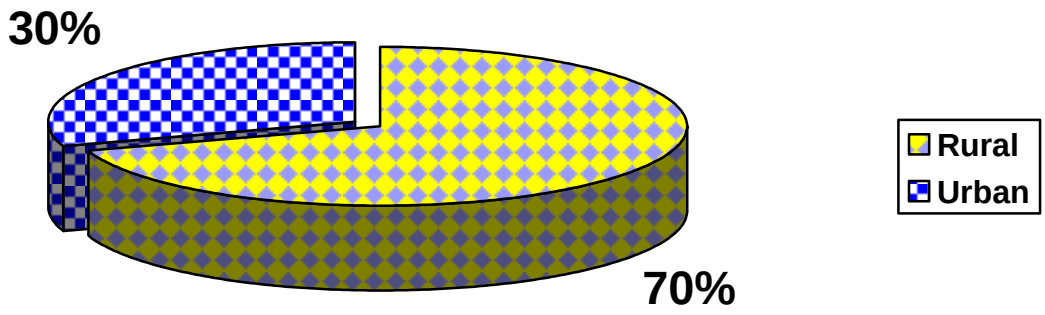
	No. of cases	**Percentage**
Male	54	90%
Female	6	10%
Total	**60**	**100%**

From the above table, it is observed that the majority of the patients in this present study were belong to male sex.

The male to female ratio was 9:1.

PIE DIAGRAM SHOWING GEOGRAPHIC DISTRIBUTION

OF CASES



This diagram shows 70% of cases were rural people.

Table showing geographical distribution

GEOGRAPHICAL DISTRIBUTION: Table – 3				
Area	**No. of cases**	**Male**	**Female**	**Percentage**
Rural	42	37	5	70%
Urban	18	17	1	30%
Total	60	54	6	100%

It is observed from the above table 42 out of 60 cases were from the rural areas constituting 70% of the total cases. 18 cases from the urban area constituted only 30% of the total.

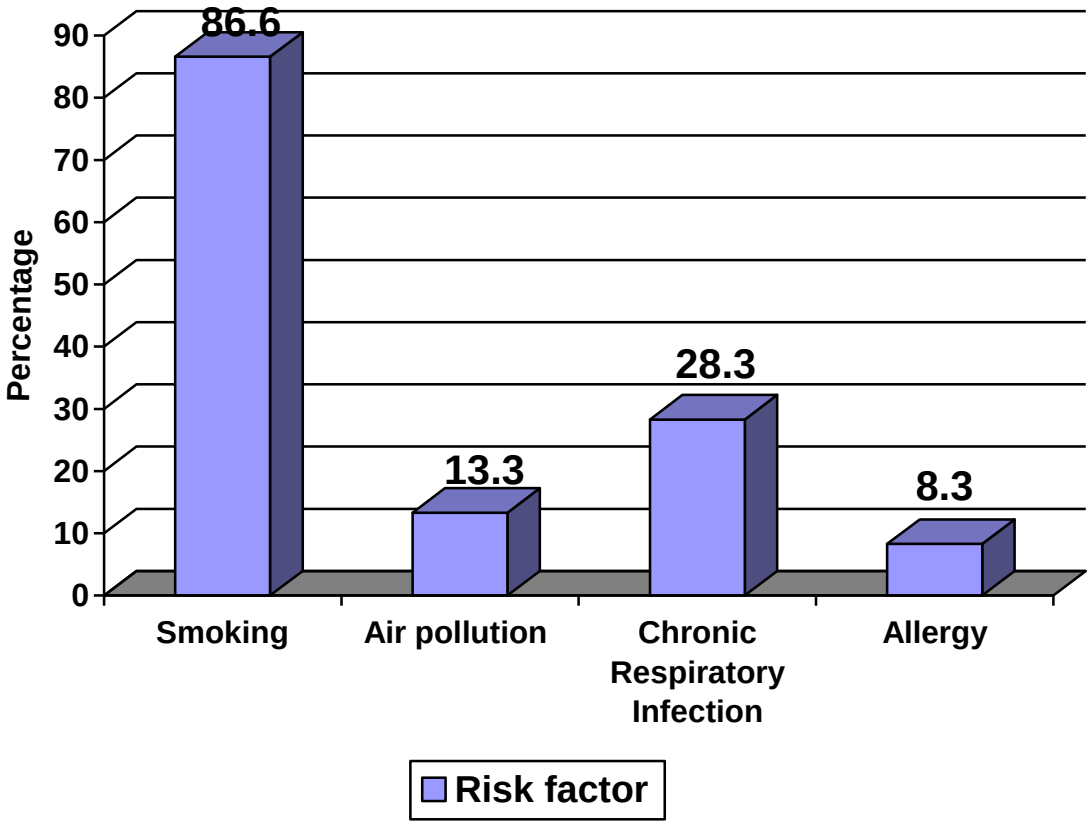
RISK FACTOR DISTRIBUTION

Table -4

Risk factor		**Male**	**%**	**Female**	**%**	**Total**	**Percentage**
Smoking	Active	50	92.5%			52	86.6%
	Passive			2*	33.3%		
Air pollution		6	11.1%	2	33.3%	8	13.3%
Chronic Respiratory Infection		14	25.9%	3	50%	17	28.3%
Allergy		4	7.4%	1	16.6%	5	8.3%

(* Life partners of this two female patients were heavy smokers more than 20 pack-years of smoking).From the above table it was noted that major risk factor for COPD in males constituted 92.5% in males and 86.6% of total cases.

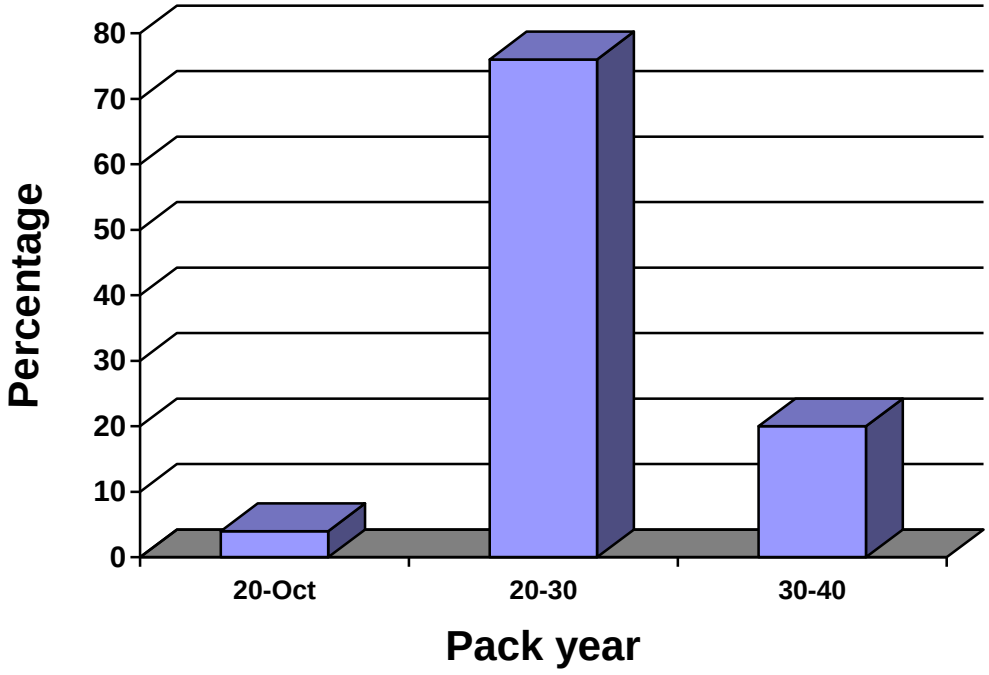
BAR DIAGRAM SHOWING RISK FACTORS IN CASES



Chronic respiratory inspection which constituted 50% of risk factor in females and 28.3% of total cases.

Air pollution constituted 13.3% of risk factor followed by allergy with constituted 8.3%.

BAR DIAGRAM SHOWING NO. OF CASES IN PACK YEAR OFSMOKING



this diagram outlays the distribution of pack years.

PACK YEARS INTENSITY OF SMOKING

Table – 5

Pack year	No. of patients	Percentage
10 – 20	2	4%
20 – 30	38	76%
30 – 40	10	20%

From the above table it can be observed that the majority of patients had more than 20 pack years of smoking which constituted 96% of total cases.

All the patients in this table were active smokers and all the patients were male.

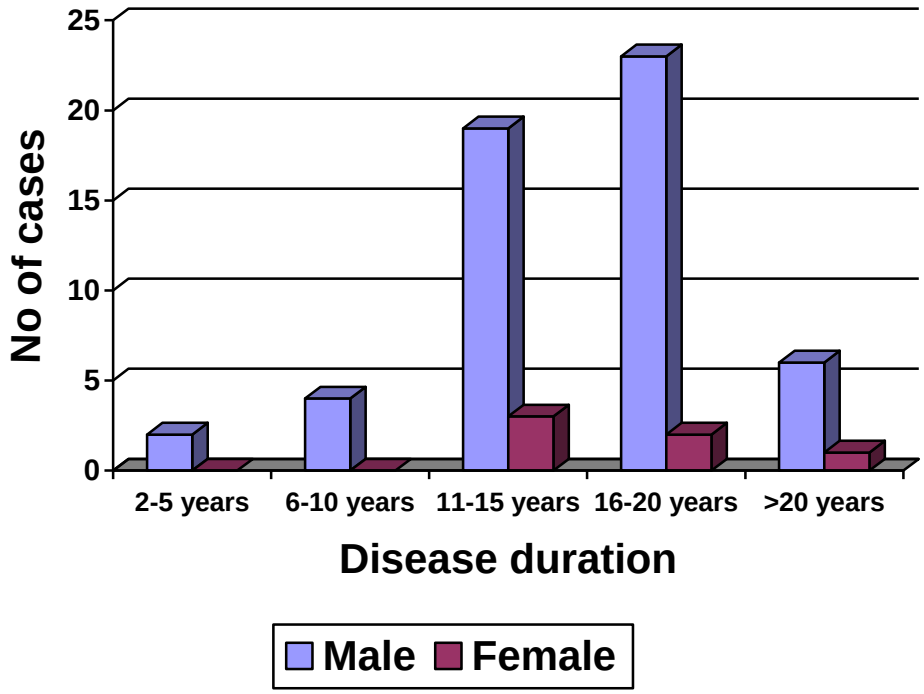
DURATION OF DISEASE

Table – 6

Disease Duration	Males	Females	Total	Percentage
2– 5 years	2	-	2	3.3
6 –10 years	4	-	4	6.6
11– 15 years	19	3	22	36.6
16– 20 years	23	2	25	41.6
> 20 years	6	1	7	11.6
Total	**54**	**6**	**60**	**100%**

From the above table it is observed that majority of patients had more than 10 years duration of disease.

DURATION OF DISEASE



Maximum number of cases were seen 16 – 20 years du ration of disease constituting 41.6% of cases.

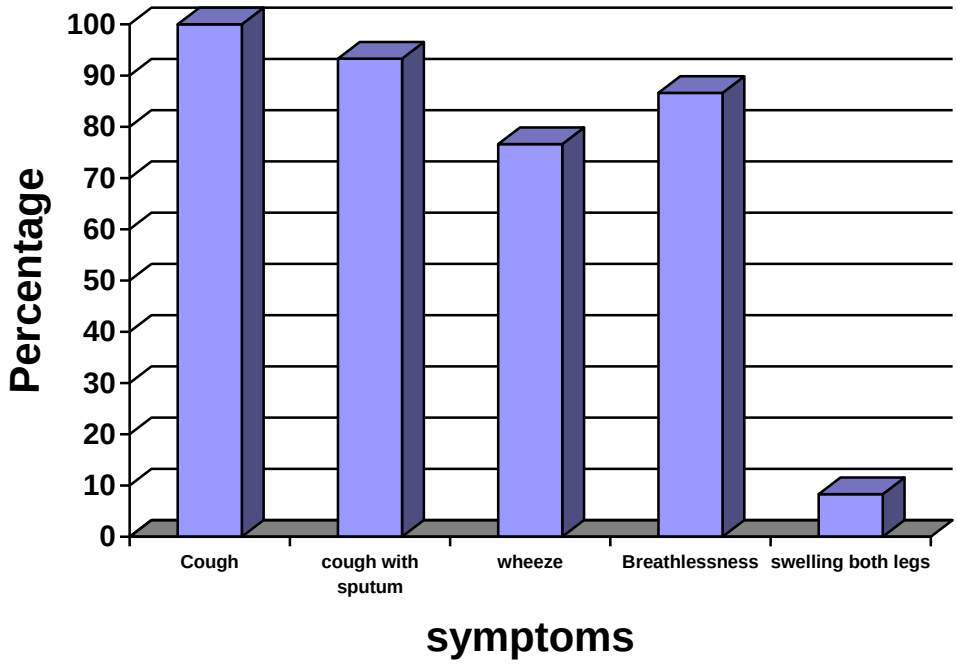
DISTRIBUTION OF SYMPTOMS

Table – 7

Symptoms	No of patients	Percentage
Cough	60	100%
Cough with expectoration of sputum	56	93.3%
Wheeze	46	76.6%
Breathlessness	52	86.6%
Swelling both legs	5	8.3%

From the above table it is noted that all the patients in this study had cough. Cough was the major symptom constituted 100% in this study.

BAR DIAGRAM SHOWING CLINICAL SYMPTOMS IN PERCENTAGE



Cough with expectoration of sputum was present in 93.3% of cases.Breathlessness which constituted 86.6% of cases.Wheeze which constituted 76.6% of cases.

Swelling of both legs were observed in 8.3% of cases

.

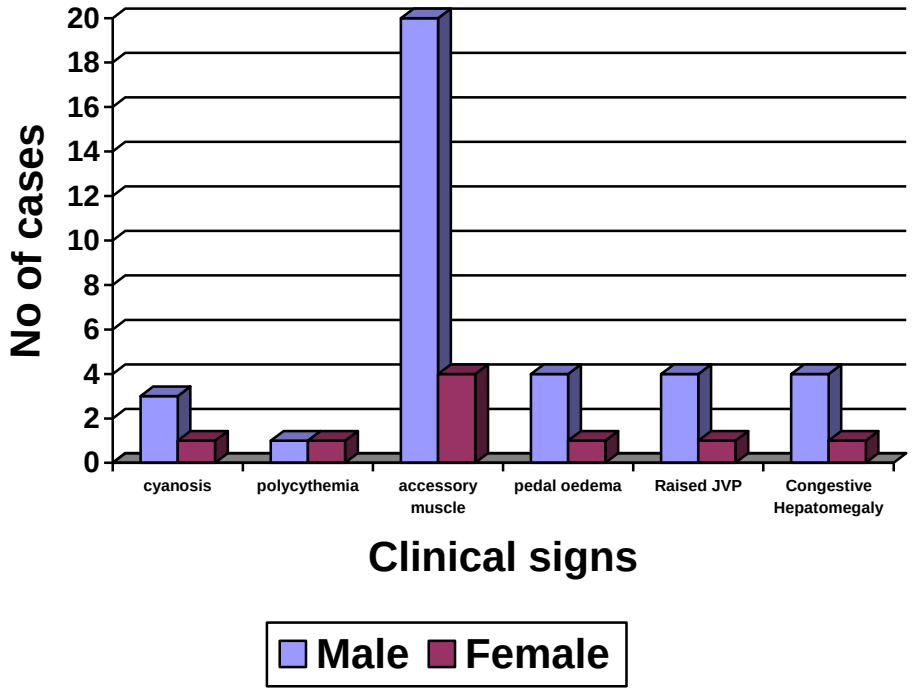
DISTRIBUTION OF CLINICAL SIGNS

Table – 8

Clinical signs	No. of patients	Male	Female	Percentage
Cyanosis	4	3	1	6.6%
Polycythemia	2	1	1	3.3%
Accessory muscles working	24	20	4	40%
Pedal oedema	5	4	1	8.3%
Raised JVP	5	4	1	8.3%
Congestive Hepatomegaly	5	4	1	8.3%

From the above table it is observed that active accessory muscles of respiration (inter costal in drawing) was the major clinical sign observed in 40% of the cases.

BAR DIAGRAM SHOWING CLINICAL SIGNS IN PERCENTAGE



In this study polycythemia was observed in 3.3% of cases.Pedal oedema, Raised JVP and Congestive Hepatomegaly were observed in 8.3% of cases.

DISTRIBUTION OF RADIOLOGICAL FINDINGS

Table – 9

Chest x-ray	No. of patients	Percentage
Low flattened diaphragm with hyperinflated lungs	39	65
Obtuse costophrenic angle	30	50
Reduction in no and size of pulmonary vessels in periphery	12	20
Normal	12	20

From the study it is observed that Low flattened diaphragm with hyperinflated lungs was the major radiological feature present in 65% of cases.

50% of cases were shown obtuse costo phrenic angle in chestx-ray.

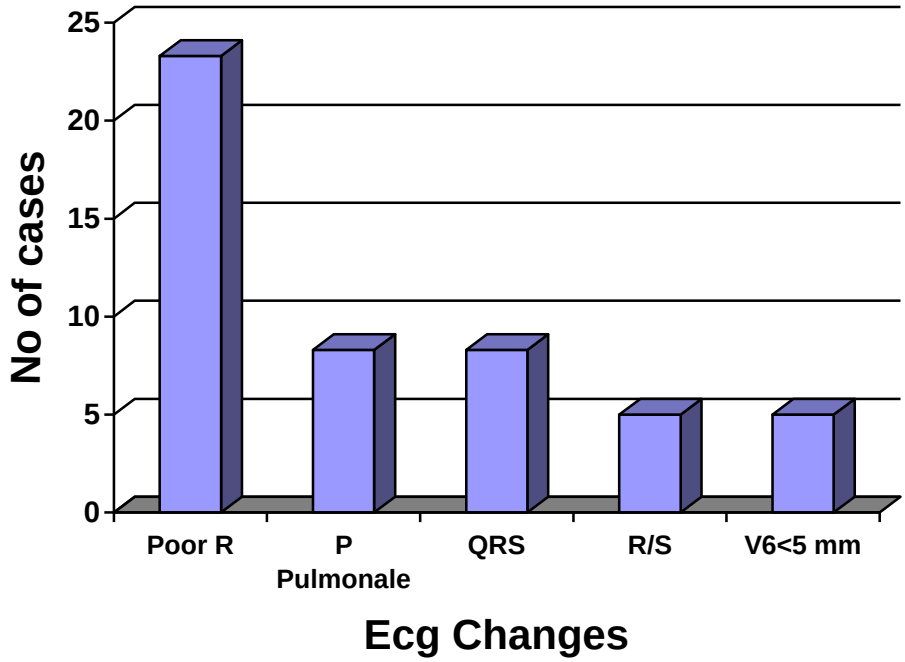
20% of the patients in this study had Normal x-ray chest.

DISTRIBUTION OF ECG CHANGES IN VARIOUS STAGES OF COPD

Stage of (*GOLD criteria*)	**ECG changes**					
	Poor 'R' Wave progression in $V_1 - V_6$	**'P' Pulmonale**	**QRS > + 90^0 in frontal plane**	**R/S < 1 in V_5 V_6**	**V_6< 5 mm**	**Total**
Stage I	-	-	-	-	-	-
Stage II	3	-	-	-	-	3
Stage III	5	0	0	-	-	5
Stage IV	6	5	5	3	3	22
Total	**14**	**5**	**5**	**3**	**3**	**30**

From the above table it is observed that more number of ECG changes were seen in patients in Stage IV of COPD.

BAR DIAGRAM SHOWING ECG CHANGES IN COPD



There was no ECG changes observed in patients with Stage I of COPD.

DISTRIBUTION OF ECG CHANGES

Table – 11

ECG changes	Male	Female	Total	Percentage
Poor 'R' Wave progression in $V_1 - V_6$	12	2	14	23.3%
'P' Pulmonale	4	1	5	8.3%
QRS > + 90^0 in frontal plane	4	1	5	8.3%
R/S < 1 in V_5 V_6	2	1	3	5%
V_6< 5 mm	2	1	3	5%

From the above table it is observed that most frequent ECG abnormality in this study was poor progression of 'R' wave which was observed in 23.3% of cases.

8.3% of cases showed evidence of cor pulmonale like 'P' Pulmonale and right axis deviation.

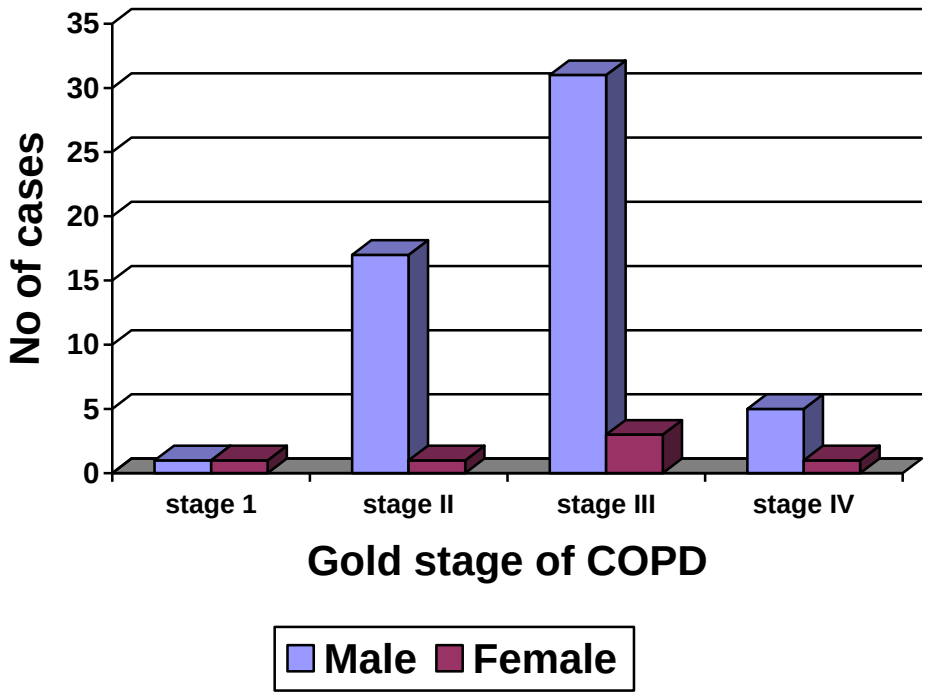
DISTRIBUTION OF CASES IN GOLD STAGING OF COPD

Table – 12

GOLD stage of COPD	Severity	No. of cases			Percentage
		Male	Female	Total	
Stage I $FEV_1 \geq 80\%$	Mild	1	1	2	3.33%
Stage II Fev_1 50% – 80%	Moderate	17	1	18	30%
Stage III FEV_1 30% – 50%	Severe	31	3	34	56.6%
Stage IV $FEV_1 < 30\%$	Very Severe	5	1	6	10%

From the above table, majority of the patients in the study were Gold Stage III of COPD showing severe airflow obstruction (FEV_1 30% - 50%).

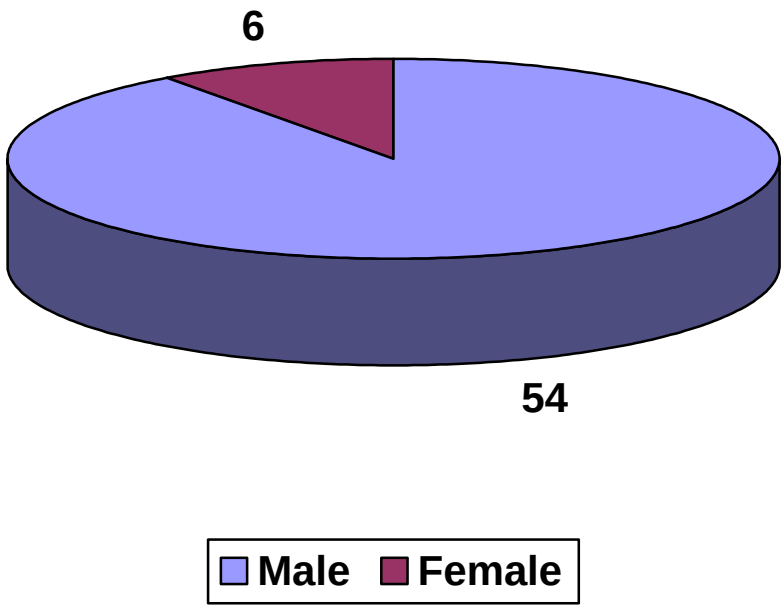
BAR DIAGRAM SHOWING GOLD STAGE OF COPD CASES



10% of the patients showed FEV_1< 30% very severe air flow obstruction belonged to Stage IV of COPD.

PIE DIAGRAM SHOWING CLINICAL SIGNS OF RHF IN COPD

CASES IN PERCENTAGE



Right heart failure was predominantly seen in male population which has the higher prevalence of COPD .

DISTRIBUTION OF RIGHT HEART FAILURE IN COPD

Table – 13

Total No. of patients			Patient with clinical signs of Right Heart Failure			Percentage
Male	**Female**	**Total**	**Male**	**Female**	**Total**	
54	6	60	4	1	5	8.33 %

From the above table it is observed that 5 cases having the clinical features of right heart failure constituting 8.33% of total.

All the 8.33% are also proved by echocardiography.

DISTRIBUTION OF RIGHT HEART FAILURE IN DURATION OF DISEASE

Table - 14

Duration of disease	No. of cases	
	Male	Female
2 – 5 years	-	-
6 –10 years	-	-
11 – 15 years	-	-
16 – 20 years	1	-
> 20 years	3	1
Total	4	1

From the above table it is observed more number of patients with right heart failure having disease more than 20 years of duration constituting 80% of the total cases of right heart failure in COPD.

DISCUSSION

1. Total number of cases were 60 in this study.

AGE AND SEX:

2. In the present study maximum number of cases were in the age group of 61-70 years. This coincides well with the various study as follows,

   i) Wig K.L Guleira K. S et al 1964 study showed maximum number of cases in 55-65 years. [20]

   ii) Higham MA Dawson et al at 2001 showed maximum number cases in 61-70years. [52]

   iii) Suzzane Hurd et al showed maximum number of cases in 60-70 years. [4]

3. In this study, it was noted the incidence of COPD was higher in males than females with male to female ratio of 9:1.

   Male cases accounted for 90% of the total cases in this study. This finding also coincides with the following studies,

   1. Trivedi H.S. et al study showed 80% of cases were male. [46]
   2. Mieguere et al showed 93% of cases were male. [44]

GEOGRAPHICAL DISTRIBUTION:

In this study it is observed that 70% of patients were from rural areas and 30% were from urban areas.

The study by Bhattacharya S.N. et al, 1975, also made similar observation. [21]

Anderson et al, 1963, made similar observation in his study. [54]

In spite of heavy Air pollution, the urban areas contributed only 30% of the cases in this study.

RISK FACTORS:

Among the various risk factors Smoking is the major risk factor accounted for 87% of the causative risk factor in this present study.

Ashley F, Kannel WB et al, 1975, made similar observation. [54]

Burrows et al, 1979, made similar observation. [25]

Intensity of smoking was expressed in pack-years in this study. As the number of pack-years more than 20, increased predisposition to COPD was observed in many studies.

In this study, most of the patients were 20-30 pack-years of smoking constituting 96% of the total.

Higgins ITT et al, 1959, observed in his study 86% patients were more than 30 pack-years of smoking. [55]

Chronic respiratory infection which constituted 28.3% was the second major risk factor observed in this study, followed by Air Pollution and

Allergy.

Burrows B et al, 1977, also made similar observation.In his study Chronic respiratory infection constituted 35%. [56]

Air pollution constituted 13.3% of risk factor and Allergy 8.3% of risk factors. [24]

DURATION OF DISEASES:

In this present study of most of the cases were above 10 years of duration of the disease.

Barnes BJ et al, 1999, in his study observed 55% had more than 15 years. [57]

Symptoms:

In this study cough and cough with expectoration of sputum was observed in 100% and 93% of cases respectively

Burrows et al, 1979, made in his study 96% of patients had cough with exportation of sputum. [25]

Dyspnoea was observed in 86.6% cases.

Altose MD et al, 1985, observed 90% of cases had dyspnoea. [58]

Physical signs:

This study observed acting accessory muscles of respiration was the major physical sign observed in 40% of cases.

Polycythemia (Secondary to hypoxia) was observed in only 3.3% ofcases.

Pedal oedema raised JVP and congestive hepatomegaly observered in 8.3% of cases.

Chest X-ray PA view:

In this present study most common radiological finding in chest , X-ray PA view was hyper inflated lungs with low flattened diaphragm which constituted 65% of cases.

Thurlbeck WM et al, 1970, observed in his study 78% of the patients showed radiological evidence of emphysema with chronic bronchitis. [59]

Normal X-ray chest was observed in 20% of cases.

Electrocardiography:

By electrocardiography poor progression of 'R' Wav e was the most frequent abnormality detected in this present study constituted 23.3%.

8.3% of cases showed 'P' pulmonale and QRS axis > + 90^0

R/S < 1 in V_5 -V_6 observed in 5% of cases.

R wave in V_6< 5 mm was observed in 3.3% of cases.

Boushy SF et al., 1971, in his study observed that 'P' pulmonale and QRS axis > + 90^0 were the major ECG changes present in 12.5% of the patient. [10]

It was observed from this study more number of cases with ECG changes were seen in Stage IV COPD. Which denoted that as the severity of COPD increases, ECG changes also increases.

SPIROMETRY:

In the study most number of patients were in GOLD Stage III COPD which constituted 56.6% of cases. The study by Higham et al [52] showed that majority of patients were in Stage III (BTS Scheme for COPD) constituted 57 – 58% of cases.

Renzeti AD et al, 1966 observed 76% of his cases belong to moderate to severe stages of COPD. [49]

Right Heart Failure:

In this study it is observed that 8.3% of cases showed clinical evidence of right heart failure. All the patients who showed the clinical evidence of right heart failure were subjected to echocardiography and confirmed the presence of right heart failure.

Mattay R et al, 1981, observed that 12.5% of his cases were showed evidence of cor pulmonale. [16]

SUMMARY

In this study on Chronic Obstructive Pulmonary Diseases the following facts were observed.

COPD is the disease of aged as evidenced by majority of patients in the present study belong to the age group of 50 – 80 years.

COPD has male predominance as evidenced by 9:1 ratio of Male to Female due to high prevalence of smoking habits observed in males.

Cigarette smoking was the major risk factor for COPD in this study.

Cough / Cough with expectoration of sputum was the major clinical symptom observed in this study.

Acting accessory muscles of respiration with pursed lip breathing was the major clinical sign observed in this study.

Spirometry is the mandatory investigation to diagnose and assess the severity of COPD.

Most number of cases had severe airway obstruction which was not reversible.

High flattened diaphragm and hyper lucent lungs were the most common chest x-ray finding observed in this study.

Poor progression of ‘R’ wave in chest leads, P Pulmonale, QRS > + 90^0, R wave in V_6< 5mm and R/S < 1 in V_5 V_6 were the ECG changes observed in this study.

The clinical incidence of Right heart failure in COPD in this study was 5% which was confirmed by ECHO.

CONCLUSION

1. Chronic Obstructive Pulmonary Diseases is a preventable disease as smoking is the major risk factor for Chronic Obstructive Pulmonary Diseases.

2. Spirometry is mandatory to diagnose and assess the severity of Chronic Obstructive Pulmonary Diseases

3. FEV_1 was the single most important parameter in spirometry to diagnose Chronic Obstructive Pulmonary Diseases along with the less than 15% of reversibility of airflow obstruction to bronchodilators.

4. Severity of Chronic Obstructive Pulmonary Diseases has direct relation with incidence of ECG changes in Chronic Obstructive Pulmonary Diseases.

5. Clinical signs of right heart failure in Chronic Obstructive Pulmonary Diseases were effective in screening the patients for cor pulmonale.

6. Right heart failure denotes severity and duration of Chronic Obstructive Pulmonary Diseases.

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CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR. SUGUMAR.T,** Post graduate in the Department of General Medicine, Thanjavur Medical College & Hospital, Thanjavur – 613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations.

Place :

Date :

PROFORMA

STUDY ON COPD

NAME: **AGE:** **SEX:M/F**

I.P / O.P.

ADDRESS: **NO.:**

OCCUPATION: **ECONOMY CLASS:** High

Middle

Low

DURATION OF DISEASE

PRESENTING SYMPTOMS:

- Cough
- Cough with expectoration of sputum
- Breathlessness
- Wheeze
- Chest pain
- Fever
- Haemoptysis
- Both Legs Swelling
- Others (Specify)

RISK FACTORS AND TRIGGERS:

- Smoking
- Air Pollution like, Smoke / Fumes / Dust
- Recurrent respiratory infection.
- Atopy and Allergy

SMOKING HISTORY: ♦ Active ♦ Passive

Active: ♦ Cigarette / Ciger / Beedi

♦ Age at smoking started.

♦ Intensity of smoking in Pack-years.

Passive: ♦ Father / Husband / Son / Other (Specify Relation)

♦ Pack-year of smoking

Family History: (A) Bronchial asthma **(B)** COPD

OTHER DISEASES:

♦ Pulmonary TB ♦ CAHD ♦ HT ♦DM ♦ Valvular Heart disease

GENERAL EXAMINATION:

Clinical signs:

♦ Anemia ♦ Polycythemia ♦ Cyanosis

♦ Accessory muscles of respiration active / normal

♦ Pursed lip breathing present / absent

♦ Lymph nodes ♦ Pedal oedema ♦ JVP

Vital signs:

PR: BP: RR:

Examination of systems:

RS: Inspection

Palpation

Percussion

Auscultation

CVS:

P/A: Congestive hepatomegaly present / absent

Lab values

i) Alb. **ii)** Sugar

iii) DC **iv)** Hb%

iv) Urea

Chest X ray PA view

GOLD Stage	**FEV_1**	**FEV_1 / FVC**
Stage I		
Stage II		
Stage III		
Stage IV		

i) Normal

ii) Poor progression of ‘R’ wave in chest leads

iii) ‘P’ Pulmonale

iv) QRS > + 90^0

v) R / S in V_5 - V_6< 1

vi) V_6 R < 5mm

♦ Echo:

KEY TO MASTER CHART

i. **P / Y –** Pack Years

ii. **C.R.I. –** Chronic Respiratory Infection

iii. **SYMPTOMS**

a – Cough

b – Cough with expectoration of sputum

c – Wheeze

d – Breathlessness

e – Both Legs swelling

iv. **SIGNS**

a – Cyanosis

b – Polycythemia

c - Accessory muscles working

d - Pedal oedema

e - Raised JVP

f - Congestive Hepatomegaly

v. **ECG CHANGES**

a – Poor 'R' Wave progression in V_1– V_6

b.- ' P'Pulmonale

- QRS > + 90^0in frontal plane

d.- R/S < 1 in V_5V_6

e. **-** V_6< 5

MASTER CHART

S.No	I.P / O.P.No.	NAME	AGE	SEX	RISK FACTORS					SYMPTOMS					SIGNS						R / U	SPIROMETRY FINDINGS				DISEASE DURATION	ECG CHANGES						RHF FEATURES
					SMOKING		C.R.I	ALLERGY	AIR POLLUTION	a	b	c	d	e	a	b	c	d	e	f		I	II	III	IV		a	b	c	d	e	NORMAL	
					ACTIVE in P/ Y	PASSIVE																											
1	38838	R. Natarajan	47	M	-		-	+	-	+	-	-	-	-	-	-	-	-	-	-	U		+			11						+	
2	38846	S. Mohan	70	M	32.5		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		17						+	
3	38854	K. Kupusamy	68	M	37.5		+	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		20	+					-	
4	38862	R. Danabalan	55	M	20.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+		+	12						+	
5	38870	S. Cheliyan	72	M	28.5		+	-	-	+	+	-	+	-	-	-	+	-	-	-	R			+		22	+					-	
6	38878	A. Savithiri	61	F			+	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		15						+	
7	38886	P. Arunachalam	65	M	24.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		16						+	
8	38892	G. Ganesan	52	M	22.5		-	-	-	+	+	-	+	-	-	-	+	-	-	-	R			+		14						+	
9	38900	A. Mansoor	62	M	23.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		16						+	
10	38837	A. Gabriel	66	M	35		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		19						+	
11	38846	S. Nagarajan	55	M	25.5		-	-	+	+	+	+	+	-	-	-	-	-	-	-	R		+			15						+	
12	38953	A. Chokkalingam	67	M	27.5		-	-	-	+	+	+	+	-	-	-	+	-	-	-	U			+		17						+	
13	38761	R. Pakkirisamy	56	M	26.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			13						+	
14	39677	A. Velusamy	68	M	28		-	-	-	+	+	-	+	-	-	-	-	-	-	-	U			+		18						+	
15	38869	C. Abdulrahman	57	M	27.5		-	-	-	+	+	+	-	-	-	-	-	-	-	-	R		+			17						+	
16	38633	S. Rani	68	F			+	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		16						+	
17	39741	M. Ramesh	61	M	22		-	+	-	+	-	+	-	-	-	-	-	-	-	-	R			+		13						+	
18	40849	K. Dharman	51	M	21.5		-	-	-	+	+	-	-	-	-	-	-	-	-	-	U		+			12						+	
19	41946	A. Kaliyan	41	M			-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		16						+	
20	41989	C. Jayakumar	52	M	22.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			14						+	
21	42004	B. Madasamy	77	M	39		+	-	-	+	+	-	+	+	+	-	+	+	+	+	R				+	25	+	+	+	+	+	-	+
22	42353	S. Chellaiyan	63	M	23.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		18						+	
23	42463	M. Kumarasamy	64	M	26		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		17						+	
24	42764	L. Arulkumar	74	M	29		+	-	-	+	+	+	+	-	-	+	+	-	-	-	R		+		+	19	+					+	
25	42981	C. Kunjamal	53	F		+	+	-	-	+	+	-	+	-	-	-	-	-	-	-	R			+		13	+					+	
26	42999	T. Kulanjinathan	56	M	22.5		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		18						+	
27	43401	A. Mathews	79	M	38.5		-	-	-	+	+	+	+	+	-	+	+	+	+	+	R				+	23	+	+	+	+	+	-	+
28	43589	M. Anwarbasha	65	M	26.5		-	-	-	+	+	-	+	-	-	-	-	-	-	-	R			+		20						+	
29	43691	K. Ramaiyan	42	M	22		+	-	-	+	+	+	+	-	-	-	+	-	-	-	U			+		6						+	
30	43786	A. Periyasamy	72	M	28		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		17	+					+	

MASTER CHART

S.No	I.P / O.P.No.	NAME	AGE	SEX	RISK FACTORS					SYMPTOMS					SIGNS						R / U	SPIROMETRY FINDINGS				DISEASE DURATION	ECG CHANGES						RHF FEATURES
					SMOKING		C.R.I	ALLERGY	AIR POLLUTION	a	b	c	d	e	a	b	c	d	e	f		I	II	III	IV		a	b	c	d	e	NORMAL	
					ACTIVE in P/ Y	PASSIVE																											
31	44301	M. Thippusulthan	67	M	25		-	+	+	+	+	+	+	-	-	-	-	-	-	-	U		+			15						+	
32	44672	S. Selladurai	77	M	25.5		+	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		16						+	
33	44963	S. Chinnakannu	47	M	26		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		12	+					-	
34	45002	A. Samson	66	M	25.5		+	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+		+	18						+	
35	45146	S. Asokan	55	M	25		-	-	+	+	+	+	+	-	-	-	+	-	-	-	R			+		14	+					-	
36	45293	L. Malini	55	F			-	+	+	+	-	+	+	-	-	-	-	-	-	-	R	+		+		13						+	
37	45300	R. Sundaram	46	M			-	-	+	+	+	+	+	-	-	-	-	-	-	-	U			+		17						+	
38	45444	R. Gopalan	68	M	28		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		16						+	
39	45498	M. Kaliraj	35	M			-	+	-	+	+	+	+	-	-	-	-	-	-	-	R			+		12						+	
40	46501	S. Arulapan	63	M	20		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		12						+	
41	46798	U. Nageshan	72	M	34.5		-	-	-	+	+	+	+	+	+	-	+	+	+	+	R					21	+	+	+	-	-	-	+
42	46832	S. Mani	63	M			-	-	-	+	+	+	+	-	-	-	+	-	-	-	U			+		18						+	
43	46946	R. Sundaraman	62	M	35		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R		+			15						+	
44	47005	P. Ashwinth	34	M	14		-	-	+	+	+	+	+	-	-	-	-	-	-	-	U	+		+		3						+	
45	47142	V. Kalimuthu	36	M	16		-	-	-	+	+	+	-	-	-	-	-	-	-	-	R		+			4						+	
46	47237	R. Lakshmi	73	F		+	+	-	-	+	+	-	+	+	+	-	+	+	+	+	R			+		21	+	+	+	+	+	-	+
47	47252	R. Arumugam	62	M	32.5		+	-	-	+	+	+	-	-	-	-	-	-	-	-	R			+		7						+	
48	47449	K. Rajavel	52	M	26		-	-	-	+	-	-	-	-	-	-	-	-	-	-	U		+			7						+	
49	47501	K. Sundarajan	66	M	25		-	-	-	+	+	+	+	-	-	-	+	-	-	-	R			+		17						+	
50	47649	R. Elavarasi	53	M			-	-	+	+	+	+	+	-	-	-	-	-	-	-	R		+			11						+	
51	47733	M. Youniskhan	52	M	24		+	-	-	+	+	-	+	+	+	-	+	+	+	+	R				+	12						+	
52	47777	P. Karuppiah	55	M	23		-	-	-	+	+	+	+	-	-	-	-	-	-	-	U			+		14						+	
53	47904	P. Vasu	74	M	35		+	-	+	+	+	+	+	-	-	-	-	-	-	-	R			+		25						+	
54	48101	M. Sarangi	76	M	30		+	-	-	+	+	-	+	-	-	+	+	+	+	+	R		+		+	24	+	+	+	-	-	-	+
55	48203	M. Ayyasamy	63	M	24		-	-	-	+	+	-	+	-	-	-	-	-	-	-	R			+		15	+					+	
56	48346	R. Kannan	55	M	27		-	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		16						+	
57	48647	A. Ramasamy	56	M	26.5		+	-	-	+	+	+	+	+	-	+	+	-	-	-	R				+	19						+	
58	48999	M. Chellakannu	64	M	23		-	-	-	+	+	-	+	-	-	-	-	-	-	-	R			+		18						+	
59	49233	P. Selvam	56	M	28		-	-	-	+	+	+	+	-	-	-	+	-	-	-	U			+		8						+	
60	49435	S. Varathan	59	M	24.5		+	-	-	+	+	+	+	-	-	-	-	-	-	-	R			+		12						+	